any reaction, and yet recovery occurred. Two of his cases presented the symptoms of acute anterior poliomyelitis.

Suckling[1] reports a case in which paraplegia occurred twice, each time a fortnight after an attack of ague, and in each attack recovery commenced on the third morning after the onset of the paraplegia, and was complete in a few hours.

Suckling refers to a case reported by Romberg as far back as 1853. The attacks of paraplegia occurred suddenly on alternate mornings at the same hour and passed off in a few hours. Sensation was unaffected, but the sphincters were paralyzed. The attacks were soon checked by quinine. Suckling refers to similar cases reported by Harting and Erb.

A case of malarial hemiplegia, in which quinine had a remarkably beneficial effect, is reported by Pascal,[2] but no necropsy was obtained. Cases of malarial paralysis, both motor and sensory, but without necropsy, have been reported by Stockwell.[3] One of malarial paraplegia, without necropsy, is recorded by Hurd.[4] Boinet and Salebert give references to twenty-four papers in which motor disturbances of malarial origin are described, and at the Eleventh International Congress Boinet[5] said he had seen many motor disturbances in malaria, such as paralyses, convulsions, and choreiform movements. Csillag[6] also has reported cases of malarial paralysis, in some of which recovery occurred after the administration of quinine.

Laveran,[7] in his recent monograph on malaria, refers to many papers in which paludism is described as being complicated by paralysis of spinal or cerebral origin, and says that he has observed these paralyses several times. He refers to two cases which presented the symptoms of acute anterior myelitis and were apparently cases of malaria. He observed also a case of malarial hemiplegia, and in speaking of these malarial paralyses he says that they are sometimes persistent and sometimes transitory.

One of the most recent works on malaria has come from the pen of Mannaberg.[8] He also refers to a number of clinical cases of malarial paralysis, and acknowledges that most of the cases of nervous disease resulting from malaria are without any study of the lesions. Neuropathologists, he says, have treated malaria in a "stepmotherly" fashion.

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[1] C. W. Suckling. Brain, 1888, vol. x., p. 474.

[2] J. Pascal. Archives de Médecine et de Pharmacie Militaires, 1887, vol. x., p. 145.

[3] G. A. Stockwell. The Medical and Surgical Reporter, March 17, 1888, p. 323.

[4] A. W. Hurd. Buffalo Medical and Surgical Journal, 1888–1899, vol. xxviii., p. 19.

[5] Boinet. Eleventh International Congress, 1894, vol. iii.; Internal Medicine, p. 82.

[6] J. Csillag. Wiener med Presse, 1895, p. 1323.

[7] A. Laveran. "Traité du Paludisme," 1898.

[8] J. Mannaberg. "Die Malaria-Krankheiten," Nothnagel's "Specielle Path. und Ther.," vol. ii., Part II.

The great majority of the cases of malaria with nervous symptoms are, according to Mannaberg,[1] due to the æstivo-autumnal variety of the parasite, and Thayer[2] says that the more severe æstivo-autumnal fevers of Rome are much more acyclical in their manifestations, tend frequently to become pernicious, and are more resistant to quinine. This form of malarial fever is chiefly characteristic of intensely malarious districts, particularly of those regions in the tropics where the pernicious fevers are common. The æstivo-autumnal form was also found by Ewing in some of his cases of implication of the nervous system in malaria, and it is this form which is present in the case H. A. In my preparations the pigment is found at the centre of the parasite, which, according to Thayer,[3] is indicative of full development of the organism. It is no exaggeration to say that every capillary of the central nervous system is filled with the organisms, so that the appearance under a low power is like that presented by injection with some black powder. The small vessels of the pia also contain the parasites.

There could be no doubt that these organisms found within the central nervous system were the malarial parasites, but the determination of the variety was more difficult, and I am, therefore, much pleased to have the opinion of Dr. Alfred Stengel and Dr. W. S. Thayer, both of whom examined my sections, that these parasites were of the æstivo-autumnal form.

The case shows that the symptoms of disseminated sclerosis may result from irritative vascular lesions, or imperfect nutrition, or poisoning of the nerve cells, without the formation of multiple sclerotic foci. Multiple sclerosis is chiefly a disease of inco-ordination. Ataxia, intention tremor, scanning speech, and nystagmus are all symptoms of inco-ordination, and it would seem that this inco-ordination may be caused either by the production of abnormal impulses from irritation, as in my case, or by the partial arrest of normally produced impulses in the multiple sclerotic areas of true insular sclerosis.

I found groups of bacilli within the vermis of the cerebellum, and only within this part. Possibly these were of post-mortem origin, but it is singular that they were seen only in the vermis. The malarial bacilli described by Klebs and Tommassi Crudeli in 1879 are not accepted at the present day as the cause of malaria.

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[1] Loc. cit.

[2] Loc. cit.

[3] Loc. cit., p. 67.